Published in Indoor Air 18(1): 72-73, 2008.

Letter to the Editor

Commentary on Predictive Models of Control Strategies Involved in Containing Indoor Airborne Infections, Indoor Air 16: 469-481.

Dear Editor,

The modeling of airborne disease transmission by Chen, Chang, and Liao (Indoor Air 16: 469-481) includes important advances. Their paper also helps to elucidate the potential benefits of control measures. However, I would like to point out some limitations that are not significantly addressed by the authors.

Neglected removal processes

The modeling of disease transmission by Chen et al. (2006) uses the Wells-Riley equation, as modified by Rudnick and Milton (2003), as a starting point. Underlying this model is a mass balance calculation of the airborne concentration of infectious agents which are present in small particles produced during coughing, sneezing and possibly other processes. The mass balance in the original Wells Riley equation accounts only for infectious agent removal from the indoor air by ventilation. Neglected removal phenomena include any natural die-off of the airborne infectious agents, natural depositional losses, e.g., from gravitational settling, and intentional removal, e.g. by means of particle filtration. The modified version of this model from Rudnick and Milton, by assuming that the inhaled concentration of infectious agent is proportional to the inhaled concentration of occupant-generated carbon dioxide[1], also neglects to account for infectious agent removal by any factor other than ventilation.

It is important to consider whether the other removal processes are significant compared to removal by ventilation. A typical volumetrically normalized removal rate by ventilation is 0.8 h^{-1} in an office and roughly 0.5 h^{-1} in a residence (Nazaroff 2004). In a health care facility, the removal rate by ventilation will often be higher, e.g., 5 h^{-1}. Little is known about the rate at which airborne infectious organisms, such as influenza virus present within naturally produced droplet nuclei, die or lose their ability to infect. We can, however, estimate rates of natural depositional losses and removal by filtration. These removal rates depend on particle size, which is not yet well characterized. Nevertheless, one estimate of the size of particles produced by coughing (after desiccation) is as follows: 50% of particles have an aerodynamic diameter between 1 and 2.9 μm with mean[2] of 2.1 μm and 50% of particles have an aerodynamic diameter between 2.9 and 5.8 μm with mean of 4.5 μm (Nicas et al, 2005). Measured indoor particle depositional loss rates for 2 μm particles tend to be between 0.4 and 3.5 h^{-1} (Lai 2002), which are clearly not negligible compare to the typical removal rates by ventilation. For 4.5 μm particles, measured depositional loss rates are even higher, between 1 and 5 h^{-1}. Thus, natural particle depositional losses must be considered to accurately model aerosol-based respiratory disease transmission. Public buildings in the U.S. also routinely recirculate indoor air through particle filters. In addition, there is a trend toward use of filters with significant particle removal

[1] because carbon dioxide removal from indoor air occurs only by ventilation
[2] diameter corresponding to the mean particle volume

efficiencies for particles larger than 1 μm in home heating and cooling systems. The resulting rate of removal of infectious particles depends on the volumetrically normalized recirculation air flow rate through the filters and the particle removal efficiency of the filters. In an office or institutional building, a typical volume-normalized filtration flow rate is 4 h^{-1} (Fisk et al. 2002). Particle removal efficiencies of filters vary widely, but will rarely be less than 0.35 for 2.5 μm particles and 0.7 for 4.5 μm particles (Fisk et al. 2002). The resulting removal rates by filtration, i.e., products of recirculation air flow rate and particle removal efficiency, are then at least 1.4 h^{-1} and 2.8 h^{-1}, for 2 and 4.5 μm size particles, respectively. Thus, removal of infectious agents by normal filtration systems also needs to be considered to accurately model respiratory disease transmission in public buildings.

Introduction of new infectious individuals

Chen et al, (2006) use their model to estimate whether a respiratory disease in a building will spread or die out, using the basic reproductive number R_o, "defined as average number of successful secondary infection cases generated by a typical primary infected case in an entirely susceptible population". If R_o is less than unity, the infection will die out. They use their model to estimate whether R_o is greater than or less than unity for a range of building conditions. However, the model applies for an isolated population[3] while the individuals in real buildings may become infected from sources outside of the building in question. In assessing the spread of disease in a community, transmission rates within a building cannot be decoupled from the transmission rates outside of the building.

Multiple mechanisms of disease transmission operating in parallel

The Wells-Riley equation, and models derived from that equation account only for infection caused by inhalation of infectious particles. However, for many respiratory infections such as those caused by influenza and human rhinovirus, it is generally believed that multiple processes of disease transmission occur in parallel (Fisk 2000). For example, in addition to the long-range aerosol-based transmission considered in the models, there is a possibility of droplet based transmission when an infected individual coughs or sneezes and expels large droplets that have momentum and impinge on the susceptible tissues, e.g., nasal mucosa, of a susceptible neighbor. Also, respiratory infections may be transmitted via direct person-to-person contact and indirect person- surface-person contact. These mechanisms of disease transmission will be linked. For example, a case of contact-based transmission may lead to a subsequent case of aerosol based transmission. By failing to account for other transmission pathways, calculations of basic reproductive number based only on a consideration of aerosol-based transmission are likely to be substantially inaccurate.

Summary

The purpose of this commentary is not to be critical specifically of the modeling of Chen et al. (2006). To the best of my knowledge, all prior modeling of indoor airborne respiratory disease transmission, including my own efforts (Fisk et al. 2005), have failed to account for some or all of the factors mentioned above. Rather, my purpose has been to point out need to develop and test substantially more sophisticated models of indoor respiratory disease transmission. The

[3] An example of an isolated population is the set of residents of in an Antarctic research station, who have no outside visitors for extended periods (Warshauer et al. 1989).

most effective strategy may be to develop semi-empirical models with structures based on first principles and model constants determined from analyses of empirical data.

William Fisk
Sr. Staff Scientist
Indoor Environment Department
Lawrence Berkeley National Laboratory
[i]

References

Chen S-C., Chang C-F., and Liao C-M. (2006) Predictive models of control strategies involved in containing indoor airborne infections. *Indoor Air* 16: 469-481

Fisk W.J. (2000) Estimates of potential nationwide productivity and health benefits from better indoor environments: an update. Chapter 4 in *Indoor Air Quality Handbook*, editers J. D Spengler, J. M. Samet, and J. F. McCarthy. McGraw Hill.

Fisk WJ, Faulkner D, Palonen J, and Seppanen O (2002) Performance and cost of particle air filtration technologies. *Indoor Air* 12(4):223-234.

Fisk WJ, Seppanen O, Faulkner D, and Huang J (2005) Economic benefits of an economizer system: energy savings and reduced sick leave. *ASHRAE Transactions* 111(2): 673-679.

Lai A.C.K. (2002) Particle deposition indoors: a review. *Indoor Air* 12: 211-214.

Nazaroff W.W. (2004) Indoor particle dynamics. Indoor Air 14(7): 175-183.

Nicas M, Nazaroff W.W., and Hubbard A. (2005) Toward understanding the risk of secondary airborne infection: emission of respirable pathogens. *Journal of Occupational and Environmental Hygiene* 2: 143-154.

Rudnick S.N. and Milton D.K. (2003) Risk of indoor airborne infection transmission estimated from carbon dioxide concentration. *Indoor Air* 13: 421-432

Warshauer D.M., Dick E.C., Mandel A.D., Flynn T. C., and Jerde R.S. (1989) Rhinovirus infections in an isolated Antarctic station. American Journal of Epidemiology 129(2): 319-340.

[i] This work was supported by the Assistant Secretary for Energy Efficiency and Renewable Energy, Office of Building Technologies Program of the U.S. Department of Energy under Contract No. DE-AC02-05CH11231.